10/634,641

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=> s e3
L1            1 CAPSAICIN/CN

=> d l1 1

L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   404-86-4  REGISTRY
CN   6-Nonenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-8-methyl-, (6E)- (9CI)
     (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   6-Nonenamide, 8-methyl-N-vanillyl-, (E)- (8CI)
CN   6-Nonenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-8-methyl-, (E)-
CN   Capsaicin (6CI)
OTHER NAMES:
CN   (E)-N-(4-Hydroxy-3-methoxybenzyl)-8-methylnon-6-enamide
CN   Axsain
CN   Capsaicine
CN   Capsin P 50
CN   Dolenon
CN   Mioton
CN   N-(4-Oxy-3-methoxybenzyl)-8-methyl-6-nonenamide
CN   NSC 56353
CN   Ratden PE 40
CN   trans-8-Methyl-N-vanillyl-6-nonenamide
CN   Zostrix
FS   STEREOSEARCH
MF   C18 H27 N O3
CI   COM
LC   STN Files:   ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*,
       BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS,
       CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU,
       DETHERM*, DIOGENES, DRUGU, EMBASE, HODOC*, HSDB*, IFICDB, IFIPAT,
       IFIUDB, IMSDRUGNEWS, IMSRESEARCH, IPA, MEDLINE, MRCK*, MSDS-OHS,
       NAPRALERT, NIOSHTIC, PHAR, PIRA, PROMT, RTECS*, TOXCENTER, USAN, USPAT2,
       USPATFULL, VETU
         (*File contains numerically searchable property data)
     Other Sources:   DSL**, EINECS**, TSCA**
         (**Enter CHEMLIST File for up-to-date regulatory information)
DT.CA  CAplus document type:  Book; Conference; Dissertation; Journal; Patent
RL.P   Roles from patents:  ANST (Analytical study); BIOL (Biological study);
       MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC
       (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses);
       NORL (No role in record)
RLD.P  Roles for non-specific derivatives from patents:  ANST (Analytical
       study); BIOL (Biological study); PREP (Preparation); RACT (Reactant or
       reagent); USES (Uses)
RL.NP  Roles from non-patents:  ANST (Analytical study); BIOL (Biological
       study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU
       (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT
       (Reactant or reagent); USES (Uses); NORL (No role in record)
RLD.NP Roles for non-specific derivatives from non-patents:  ANST (Analytical
       study); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation);
       PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES
       (Uses)

Double bond geometry as shown.
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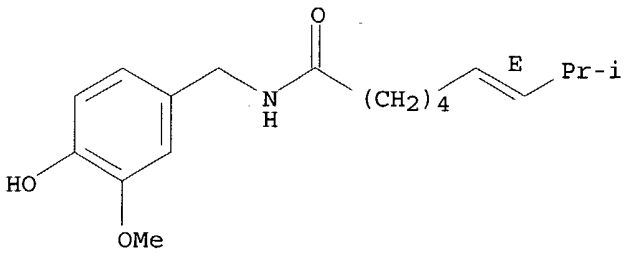
DELACROIX



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

3958 REFERENCES IN FILE CA (1907 TO DATE)
78 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
3963 REFERENCES IN FILE CAPLUS (1907 TO DATE)
45 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

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=> e myristic acid/cn
E1            1     MYRISTARGENOL B/CN
E2            1     MYRISTATE/CN
E3            1 --> MYRISTIC ACID/CN
E4            1     MYRISTIC ACID A-MONOGLYCERIDE/CN
E5            1     MYRISTIC ACID B-MONOGLYCERIDE/CN
E6            1     MYRISTIC ACID B-SITOSTERYL ESTER/CN
E7            1     MYRISTIC ACID 1-MONOGLYCERIDE/CN
E8            1     MYRISTIC ACID 2-BUTANOL ESTER/CN
E9            1     MYRISTIC ACID 2-DECANOL ESTER/CN
E10           1     MYRISTIC ACID 2-HEPTANOL ESTER/CN
E11           1     MYRISTIC ACID 2-HEXANOL ESTER, 1-METHYLPENTYL ESTER/CN
E12           1     MYRISTIC ACID 2-NONANOL ESTER/CN

=> s e3
L2            1 "MYRISTIC ACID"/CN

=> d l2 1

L2   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   544-63-8  REGISTRY
CN   Tetradecanoic acid (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Myristic acid (8CI)
OTHER NAMES:
CN   1-Tridecanecarboxylic acid
CN   Edenor C 14
CN   Emery 655
CN   Hystrene 9014
CN   Kortacid 1499
CN   n-Tetradecan-1-oic acid
CN   n-Tetradecanoic acid
CN   n-Tetradecoic acid
CN   NAA 104
CN   NAA 142
CN   Neo-Fat 14
CN   NSC 5028
CN   Philacid 1400
CN   Prifac 2942
CN   Univol U 316S
FS   3D CONCORD
DR   45184-05-2
MF   C14 H28 O2
CI   COM
LC   STN Files:   AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS,
       BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN,
       CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*,
       DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2,
       GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*,
       MSDS-OHS, NAPRALERT, NIOSHTIC, PDLCOM*, PIRA, PROMT, RTECS*, SPECINFO,
       TOXCENTER, TULSA, USPAT2, USPATFULL, VTB
         (*File contains numerically searchable property data)
     Other Sources:   DSL**, EINECS**, TSCA**
         (**Enter CHEMLIST File for up-to-date regulatory information)
DT.CA  CAplus document type:  Conference; Dissertation; Journal; Patent;
       Preprint; Report
RL.P   Roles from patents:  ANST (Analytical study); BIOL (Biological study);
       FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU
       (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT
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(Reactant or reagent); USES (Uses); NORL (No role in record)
RLD.P Roles for non-specific derivatives from patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses)
RL.NP Roles from non-patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses); NORL (No role in record)
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$HO_2C-(CH_2)_{12}-Me$

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

18134 REFERENCES IN FILE CA (1907 TO DATE)
707 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
18188 REFERENCES IN FILE CAPLUS (1907 TO DATE)
13 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

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=> e palmitic acid/cn
E1            1     PALMITELAIDOYL-COA/CN
E2            1     PALMITELAIDYLCOENZYME A/CN
E3            1 --> PALMITIC ACID/CN
E4            1     PALMITIC ACID (4-BROMOBENZOYL)METHYL ESTER/CN
E5            1     PALMITIC ACID A-MONOGLYCERIDE/CN
E6            1     PALMITIC ACID B-MONOGLYCERIDE/CN
E7            1     PALMITIC ACID 2-BUTANOL ESTER/CN
E8            1     PALMITIC ACID 2-DECANOL ESTER/CN
E9            1     PALMITIC ACID 2-HEXANOL ESTER/CN
E10           1     PALMITIC ACID 2-NONANOL ESTER/CN
E11           1     PALMITIC ACID 2-OCTANOL ESTER/CN
E12           1     PALMITIC ACID 2-PENTANOL ESTER/CN

=> s e3
L3            1 "PALMITIC ACID"/CN

=> d l3 1

L3   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   57-10-3  REGISTRY
CN   Hexadecanoic acid (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Palmitic acid (7CI, 8CI)
OTHER NAMES:
CN   1-Pentadecanecarboxylic acid
CN   Cetylic acid
CN   Edenor C16
CN   Emersol 143
CN   FA 1695
CN   Hydrofol Acid 1690
CN   Hystrene 9016
CN   Kortacid 1698
CN   Loxiol EP 278
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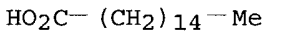
CN Lunac P 95
CN Lunac P 95KC
CN n-Hexadecanoic acid
CN n-Hexadecoic acid
CN NAA 160
CN Neo-Fat 16
CN NSC 5030
CN PA 900
CN Palmitinic acid
CN Pentadecanecarboxylic acid
CN Prifac 2960
CN Pristerene 4934
FS 3D CONCORD
DR 60605-23-4, 66321-94-6, 116860-99-2, 212625-86-0
MF C16 H32 O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PDLCOM*, PIRA, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)
DT.CA CAplus document type: Conference; Dissertation; Journal; Patent; Preprint; Report
RL.P Roles from patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses); NORL (No role in record)
RLD.P Roles for non-specific derivatives from patents: ANST (Analytical study); BIOL (Biological study); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses)
RL.NP Roles from non-patents: ANST (Analytical study); BIOL (Biological study); CMBI (Combinatorial study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses); NORL (No role in record)
RLD.NP Roles for non-specific derivatives from non-patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

37138 REFERENCES IN FILE CA (1907 TO DATE)
1324 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
37232 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

DELACROIX

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=> e stearic acid/cn
E1            1     STEAREX BEADS/CN
E2            1     STEARIC ACETIC ANHYDRIDE/CN
E3            1 --> STEARIC ACID/CN
E4            1     STEARIC ACID A-MONOGLYCERIDE/CN
E5            1     STEARIC ACID B-MONOGLYCERIDE/CN
E6            1     STEARIC ACID 1,2-PROPANEDITHIOL ESTER/CN
E7            1     STEARIC ACID 1,3-PROPANEDITHIOL ESTER/CN
E8            1     STEARIC ACID 1-MONOGLYCERIDE/CN
E9            1     STEARIC ACID 2-BUTYL ESTER/CN
E10           1     STEARIC ACID 2-HEPTANOL ESTER/CN
E11           1     STEARIC ACID 2-HEXANOL ESTER/CN
E12           1     STEARIC ACID 2-OCTANOL ESTER/CN

=> s e3
L4            1 "STEARIC ACID"/CN

=> d l4 1

L4   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   57-11-4  REGISTRY
CN   Octadecanoic acid (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Stearic acid (8CI)
OTHER NAMES:
CN   1-Heptadecanecarboxylic acid
CN   17FA
CN   400JB9103-88
CN   A 1760
CN   Adeka Fatty Acid SA 910
CN   Barolub FTA
CN   Century 1210
CN   Century 1220
CN   Century 1230
CN   Century 1240
CN   Edenor C 18/98
CN   Edenor C18
CN   Edenor HT-JG 60
CN   Edenor ST 1
CN   Edenor ST 20
CN   Emersol 120
CN   Emersol 153NF
CN   Emersol 6349
CN   F 3
CN   F 3 (lubricant)
CN   FA 1655
CN   G 270
CN   Humko Industrene R
CN   Hydrofol Acid 150
CN   Hydrofol Acid 1895
CN   Hystrene 80
CN   Hystrene 9718
CN   Hystrene 9718NF
CN   Hystrene 9718NFFG
CN   Hystrene S 97
CN   Hystrene T 70
CN   Industrene 8718
CN   Industrene 9018
CN   Industrene R
CN   Kam 1000
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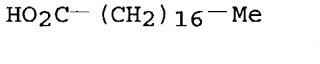
DELACROIX

CN Kam 2000
CN Kam 3000
CN Kortacid 1895
CN Loxiol G 20
CN Lunac 30
CN Lunac S 20
CN Lunac S 30
CN Lunac S 40
CN Lunac S 50
CN Lunac S 90
CN Lunac S 90KC
CN Lunac S 98
CN Lunac YA
CN n-Octadecanoic acid
CN NAA 173
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY
FS 3D CONCORD
DR 8013-28-3, 8023-06-1, 8037-40-9, 8037-83-0, 8039-51-8, 8039-52-9, 8039-53-0, 8039-54-1, 58392-66-8, 134503-33-6, 82497-27-6, 39390-61-9, 197923-10-7, 294203-07-9
MF C18 H36 O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PDLCOM*, PIRA, PROMT, PS, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, TULSA, USAN, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)
DT.CA CAplus document type: Book; Conference; Dissertation; Journal; Patent; Preprint; Report
RL.P Roles from patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses); NORL (No role in record)
RLD.P Roles for non-specific derivatives from patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses)
RL.NP Roles from non-patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses); NORL (No role in record)
RLD.NP Roles for non-specific derivatives from non-patents: ANST (Analytical study); BIOL (Biological study); CMBI (Combinatorial study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

DELACROIX

44021 REFERENCES IN FILE CA (1907 TO DATE)
2928 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
44115 REFERENCES IN FILE CAPLUS (1907 TO DATE)
19 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

DELACROIX

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=> e dohevanil/cn
E1            1      DOHEPTACONTANEDIOIC ACID/CN
E2            1      DOHEPTACONTASILOXANE(10,12)FULLEROID-C180-D6D/CN
E3            1 --> DOHEVANIL/CN
E4            1      DOHEXACONTAHECTANE/CN
E5            1      DOHEXACONTANE/CN
E6            1      DOHEXACONTANE, 2-METHYL-/CN
E7            1      DOHEXACONTANOIC ACID/CN
E8            1      DOHME/CN
E9            1      DOHMISIN/CN
E10           1      DOHNA 2E/CN
E11           1      DOHNA 2M/CN
E12           1      DOHNALIT/CN

=> s e3
L13           1 DOHEVANIL/CN

=> d l13 1

L13  ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   571203-58-2  REGISTRY
CN   4,7,10,13,16,19-Docosahexaenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-,
     (4Z,7Z,10Z,13Z,16Z,19Z)- (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   Dohevanil
FS   STEREOSEARCH
MF   C30 H41 N O3
SR   CA
LC   STN Files:   CA, CAPLUS, TOXCENTER, USPATFULL
DT.CA  CAplus document type:  Journal; Patent
RL.P   Roles from patents:  BIOL (Biological study); PREP (Preparation); USES
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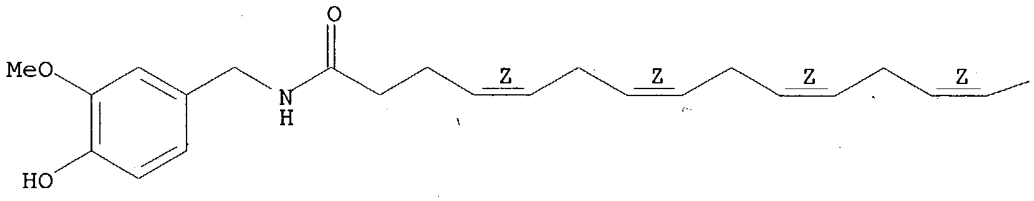
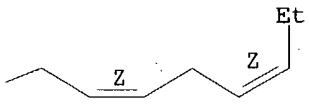
RL.NP  Roles from non-patents:  BIOL (Biological study); USES (Uses)

Double bond geometry as shown.
```

PAGE 1-A



PAGE 1-B

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**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**
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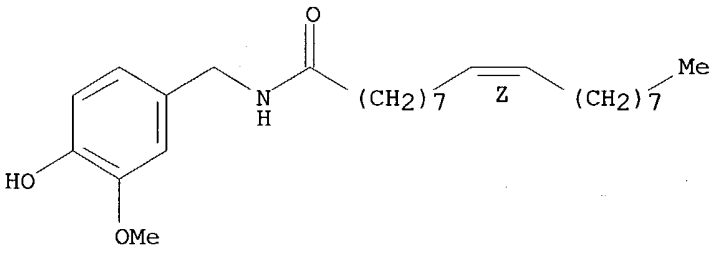
3 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> s olvanil/cn
L14 1 OLVANIL/CN

=> d l14 1

L14 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2004 ACS on STN
RN 58493-49-5 REGISTRY
CN 9-Octadecenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (9Z)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 9-Octadecenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (Z)-
OTHER NAMES:
CN N-Vanillyl oleic amide
CN N-Vanillyloleamide
CN NE 19550
CN **Olvanil**
FS STEREOSEARCH
MF C26 H43 N O3
LC STN Files: AGRICOLA, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CASREACT, CHEMCATS, CSCHEM, DDFU, DRUGU, EMBASE, IMSDRUGNEWS, IMSRESEARCH, IPA, MEDLINE, PHAR, PROMT, PROUSDDR, RTECS*, TOXCENTER, USAN, USPATFULL
(*File contains numerically searchable property data)
Other Sources: WHO
DT.CA CAplus document type: Conference; Journal; Patent
RL.P Roles from patents: BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
RL.NP Roles from non-patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses)

Double bond geometry as shown.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

78 REFERENCES IN FILE CA (1907 TO DATE)
78 REFERENCES IN FILE CAPLUS (1907 TO DATE)

DELACROIX

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E1            1     ARV-2/CN
E2            1     ARVA/CN
E3            1 --> ARVANIL/CN
E4            1     ARVELEXIN/CN
E5            1     ARVELEXINE/CN
E6            1     ARVENIN I/CN
E7            1     ARVENIN I ACETATE/CN
E8            1     ARVENIN II/CN
E9            1     ARVENIN III/CN
E10           1     ARVENIN IV/CN
E11           1     ARVENSAN/CN
E12           1     ARVENSIN/CN

=> s e3
L15           1 ARVANIL/CN

=> d l15 1

L15  ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   128007-31-8  REGISTRY
CN   5,8,11,14-Eicosatetraenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-,
     (5Z,8Z,11Z,14Z)- (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   5,8,11,14-Eicosatetraenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-,
     (all-Z)-
OTHER NAMES:
CN   Arvanil
CN   N-Vanillylarachidonamide
FS   STEREOSEARCH
MF   C28 H41 N O3
SR   CA
LC   STN Files:   BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT, CHEMCATS, CSCHEM,
       EMBASE, RTECS*, TOXCENTER, USPATFULL
         (*File contains numerically searchable property data)
DT.CA  CAplus document type:  Journal; Patent
RL.P   Roles from patents:  BIOL (Biological study); PREP (Preparation); USES
       (Uses)
RLD.P  Roles for non-specific derivatives from patents:  BIOL (Biological
       study); PREP (Preparation); USES (Uses)
RL.NP  Roles from non-patents:  BIOL (Biological study); PREP (Preparation);
       PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES
       (Uses)

Double bond geometry as shown.
```
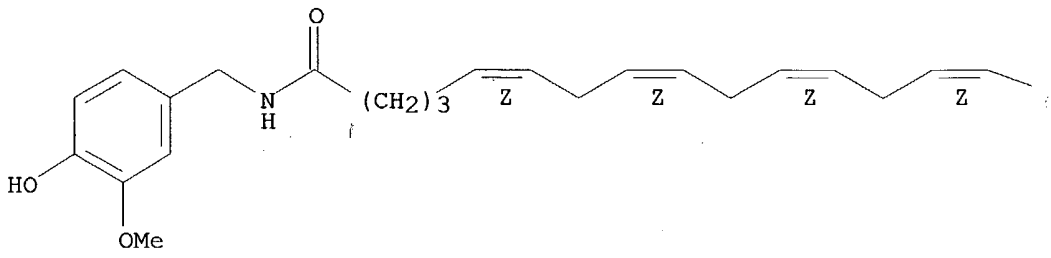
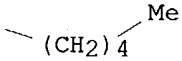

PAGE 1-A



DELACROIX



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

22 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
22 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=>

=> s l11 and (arvanil? or olvanil or docosahex?)
L12 9 L11 AND (ARVANIL? OR OLVANIL OR DOCOSAHEX?)

=> d l12 abs cbib kwic hitstr 1-9

L12 ANSWER 1 OF 9 MEDLINE on STN
AB Palmitoylethanolamide (PEA) is a bioactive **fatty acid** amide belonging to the class of N-acyl-ethanolamines (NAEs). This compound has been known since the 1950s for its anti-inflammatory effects, but was re-discovered only after the finding that another NAE, arachidonoyl-ethanolamide (anandamide, AEA), could act as an endogenous ligand of cannabinoid receptors. Although a similar function for PEA has also been proposed, this compound does not activate the two cannabinoid receptor subtypes described to date. PEA and AEA are co-synthesized by cells, and PEA might act as an 'entourage' compound for AEA, i.e. as an endogenous enhancer of AEA biological actions. Indeed, long-term treatment of human breast **cancer** cells (HBCCs) with PEA downregulates the expression of the enzyme responsible for AEA degradation, the **fatty acid** amide hydrolase, thereby leading to an enhancement of AEA-induced, and cannabinoid CB1 receptor-mediated, cytostatic effect on HBCCs. AEA is also a full agonist for the receptors of another class of bioactive **fatty acid** amides, the N-acyl-vanillyl-amines (e.g. capsaicin and **olvanil**). These sites of action are known as vanilloid receptors of type 1 (VR1). PEA enhances the VR1-mediated effects of AEA and capsaicin on calcium influx into cells. These 'entourage' effects of PEA might be attributable to modulation of VR1 activity, and could underlie the enhancement by PEA, described here for the first time, of the antiproliferative effects of VR1 receptor agonists.
2003059673. PubMed ID: 12570018. Effect on **cancer** cell proliferation of palmitoylethanolamide, a **fatty acid** amide interacting with both the cannabinoid and vanilloid signalling systems. De Petrocellis Luciano; Bisogno Tiziana; Ligresti Alessia; Bifulco Maurizio; Melck Dominique; Di Marzo Vincenzo. (Istituto di Cibernetica Eduardo Caianiello, Consiglio Nazionale delle Ricerche, Comprensorio Olivetti, Pozzuoli, Napoli, Italy.) Fundamental & clinical pharmacology, (2002 Aug) 16 (4) 297-302. Journal code: 8710411. ISSN: 0767-3981. Pub. country: England: United Kingdom. Language: English.
TI Effect on **cancer** cell proliferation of palmitoylethanolamide, a **fatty acid** amide interacting with both the cannabinoid and vanilloid signalling systems.
AB Palmitoylethanolamide (PEA) is a bioactive **fatty acid** amide belonging to the class of N-acyl-ethanolamines (NAEs). This compound has been known since the 1950s for its anti-inflammatory effects,. . . an 'entourage' compound for AEA, i.e. as an endogenous enhancer of AEA biological actions. Indeed, long-term treatment of human breast **cancer** cells (HBCCs) with PEA downregulates the expression of the enzyme responsible for AEA degradation, the **fatty acid** amide hydrolase, thereby leading to an enhancement of AEA-induced, and cannabinoid CB1 receptor-mediated, cytostatic effect on HBCCs. AEA is also a full agonist for the receptors of another class of bioactive **fatty acid** amides, the N-acyl-vanillyl-amines (e.g. capsaicin and **olvanil**). These sites of action are known as vanilloid receptors of type 1 (VR1). PEA enhances the VR1-mediated effects of AEA. . .

CT Check Tags: Female; Human; Support, Non-U.S. Gov't
Anti-Inflammatory Agents, Non-Steroidal: PD, pharmacology
*Antineoplastic Agents: PD, pharmacology
Breast Neoplasms
*Cannabinoids: ME, metabolism
*Capsaicin: AA, analogs & derivatives
Capsaicin: PD, pharmacology
Cell Division: DE, drug effects
Dose-Response Relationship, Drug
Drug Synergism
Palmitic Acids: ME, metabolism
***Palmitic Acids: PD, pharmacology**
Receptors, Cannabinoid
*Receptors, Drug: AG, agonists
Signal Transduction
Tumor Cells, Cultured
RN **404-86-4 (Capsaicin)**; 544-31-0 (palmidrol); **58493-49-5 (olvanil)**
CN 0 (Anti-Inflammatory Agents, Non-Steroidal); 0 (Antineoplastic Agents); 0 (Cannabinoids); 0 (**Palmitic Acids**); 0 (Receptors, Cannabinoid); 0 (Receptors, Drug); 0 (capsaicin receptor)

L12 ANSWER 2 OF 9 MEDLINE on STN
AB Palmitoylethanolamide (PEA) has been shown to act in synergy with anandamide (arachidonoylethanolamide; AEA), an endogenous agonist of cannabinoid receptor type 1 (CB(1)). This synergistic effect was reduced by the CB(2) cannabinoid receptor antagonist SR144528, although PEA does not activate either CB(1) or CB(2) receptors. Here we show that PEA potently enhances the anti-proliferative effects of AEA on human breast **cancer** cells (HBCCs), in part by inhibiting the expression of **fatty acid** amide hydrolase (FAAH), the major enzyme catalysing AEA degradation. PEA (1-10 microM) enhanced in a dose-related manner the inhibitory effect of AEA on both basal and nerve growth factor (NGF)-induced HBCC proliferation, without inducing any cytostatic effect by itself. PEA (5 microM) decreased the IC(50) values for AEA inhibitory effects by 3-6-fold. This effect was not blocked by the CB(2) receptor antagonist SR144528, and was not mimicked by a selective agonist of CB(2) receptors. PEA enhanced AEA-evoked inhibition of the expression of NGF Trk receptors, which underlies the anti-proliferative effect of the endocannabinoid on NGF-stimulated MCF-7 cells. The effect of PEA was due in part to inhibition of AEA degradation, since treatment of MCF-7 cells with 5 microM PEA caused a approximately 30-40% down-regulation of FAAH expression and activity. However, PEA also enhanced the cytostatic effect of the cannabinoid receptor agonist HU-210, although less potently than with AEA. PEA did not modify the affinity of ligands for CB(1) or CB(2) receptors, and neither did it alter the CB(1)/CB(2)-mediated inhibitory effect of AEA on adenylate cyclase type V, nor the expression of CB(1) and CB(2) receptors in MCF-7 cells. We suggest that long-term PEA treatment of cells may positively affect the pharmacological activity of AEA, in part by inhibiting FAAH expression.
2001439875. PubMed ID: 11485574. Palmitoylethanolamide inhibits the expression of **fatty acid** amide hydrolase and enhances the anti-proliferative effect of anandamide in human breast **cancer** cells. Di Marzo V; Melck D; Orlando P; Bisogno T; Zagoory O; Bifulco M; Vogel Z; De Petrocellis L. (Istituto per la Chimica di Molecole di Interesse Biologico, Via Toiano 6, 80072, Arco Felice, Napoli, Italy.. vdimarzo@icmib.na.cnr.it) . Biochemical journal, (2001 Aug 15) 358 (Pt 1)

249-55. Journal code: 2984726R. ISSN: 0264-6021. Pub. country: England: United Kingdom. Language: English.

TI Palmitoylethanolamide inhibits the expression of **fatty acid** amide hydrolase and enhances the anti-proliferative effect of anandamide in human breast **cancer** cells.

AB . . . either CB(1) or CB(2) receptors. Here we show that PEA potently enhances the anti-proliferative effects of AEA on human breast **cancer** cells (HBCCs), in part by inhibiting the expression of **fatty acid** amide hydrolase (FAAH), the major enzyme catalysing AEA degradation. PEA (1-10 microM) enhanced in a dose-related manner the inhibitory effect. . .

CT . . .
biosynthesis
Animals
Anti-Inflammatory Agents, Non-Steroidal: PD, pharmacology
*Antineoplastic Agents: PD, pharmacology
*Arachidonic Acids: PD, pharmacology
Blotting, Western
Bornanes: PD, pharmacology
***Breast Neoplasms: DT, drug therapy**
COS Cells
Cannabinoids: PD, pharmacology
*Capsaicin: AA, analogs & derivatives
Capsaicin: PD, pharmacology
Cell Division: DE,. . . drug effects
Cyclic AMP: ME, metabolism
Dose-Response Relationship, Drug
Endocannabinoids
Forskolin: PD, pharmacology
Glycerides: PD, pharmacology
Hydrolysis
Inhibitory Concentration 50
***Palmitic Acids: PD, pharmacology**
Protein Binding
Pyrazoles: PD, pharmacology
Receptors, Cannabinoid
Receptors, Drug: AI, antagonists & inhibitors
Reverse Transcriptase Polymerase Chain. . .

RN **404-86-4 (Capsaicin)**; 53847-30-6 (2-arachidonylglycerol); 544-31-0 (palmidrol); 60-92-4 (Cyclic AMP); 66428-89-5 (Forskolin); 94421-68-8 (anandamide)

CN 0 (Anti-Inflammatory Agents, Non-Steroidal); 0 (Antineoplastic Agents); 0 (Arachidonic Acids); 0 (Bornanes); 0 (Cannabinoids); 0 (Endocannabinoids); 0 (Glycerides); 0 (**Palmitic Acids**); 0 (Pyrazoles); 0 (Receptors, Cannabinoid); 0 (Receptors, Drug); 0 (SR 144528); 0 (**arvanil**); EC 3.5. (Amidohydrolases); EC 3.5.1.- (**fatty-acid** amide hydrolase)

L12 ANSWER 3 OF 9 MEDLINE on STN

AB We investigated the effect of changing the length and degree of unsaturation of the fatty acyl chain of N-(3-methoxy-4-hydroxy)-benzyl-cis-9-octadecenoamide (**olvanil**), a ligand of vanilloid receptors, on its capability to: (i) inhibit anandamide-facilitated transport into cells and enzymatic hydrolysis, (ii) bind to CB1 and CB2 cannabinoid receptors, and (iii) activate the VR1 vanilloid receptor. Potent inhibition of [(14)C]anandamide accumulation into cells was achieved with C20:4 n-6, C18:3 n-6 and n-3, and C18:2 n-6 N-acyl-vanillyl-amides (N-AVAMs). The

saturated analogues and Delta(9)-trans-**olvanil** were inactive. Activity in CB1 binding assays increased when increasing the number of cis-double bonds in a n-6 fatty acyl chain and, in saturated N-AVAMs, was not greatly sensitive to decreasing the chain length. The C20:4 n-6 analogue (**arvanil**) was a potent inhibitor of anandamide accumulation (IC(50) = 3.6 microM) and was 4-fold more potent than anandamide on CB1 receptors (Ki = 0.25-0.52 microM), whereas the C18:3 n-3 N-AVAM was more selective than **arvanil** for the uptake (IC(50) = 8.0 microM) vs CB1 receptors (Ki = 3.4 microM). None of the compounds efficiently inhibited [(14)C]anandamide hydrolysis or bound to CB2 receptors. All N-AVAMs activated the cation currents coupled to VR1 receptors overexpressed in Xenopus oocytes. In a simple, intact cell model of both vanilloid- and anandamide-like activity, i.e., the inhibition of human breast **cancer** cell (HBCC) proliferation, **arvanil** was shown to behave as a "hybrid" activator of cannabinoid and vanilloid receptors.

1999382278. PubMed ID: 10448105. Unsaturated long-chain N-acyl-vanillyl-amides (N-AVAMs): vanilloid receptor ligands that inhibit anandamide-facilitated transport and bind to CB1 cannabinoid receptors. Melck D; Bisogno T; De Petrocellis L; Chuang H; Julius D; Bifulco M; Di Marzo V. (Istituto per la Chimica di Molecole di Interesse Biologico, Universita di Napoli Federico II, 80131, Napoli, Italy.) Biochemical and biophysical research communications, (1999 Aug 19) 262 (1) 275-84. Journal code: 0372516. ISSN: 0006-291X. Pub. country: United States. Language: English.

AB We investigated the effect of changing the length and degree of unsaturation of the fatty acyl chain of N-(3-methoxy-4-hydroxy)-benzyl-cis-9-octadecenoamide (**olvanil**), a ligand of vanilloid receptors, on its capability to: (i) inhibit anandamide-facilitated transport into cells and enzymatic hydrolysis, (ii) bind. . . into cells was achieved with C20:4 n-6, C18:3 n-6 and n-3, and C18:2 n-6 N-acyl-vanillyl-amides (N-AVAMs). The saturated analogues and Delta(9)-trans-**olvanil** were inactive. Activity in CB1 binding assays increased when increasing the number of cis-double bonds in a n-6 fatty acyl chain and, in saturated N-AVAMs, was not greatly sensitive to decreasing the chain length. The C20:4 n-6 analogue (**arvanil**) was a potent inhibitor of anandamide accumulation (IC(50) = 3.6 microM) and was 4-fold more potent than anandamide on CB1 receptors (Ki = 0.25-0.52 microM), whereas the C18:3 n-3 N-AVAM was more selective than **arvanil** for the uptake (IC(50) = 8.0 microM) vs CB1 receptors (Ki = 3.4 microM). None of the compounds efficiently inhibited. . . Xenopus oocytes. In a simple, intact cell model of both vanilloid- and anandamide-like activity, i.e., the inhibition of human breast **cancer** cell (HBCC) proliferation, **arvanil** was shown to behave as a "hybrid" activator of cannabinoid and vanilloid receptors.

CT . . .

Line

Cell Membrane: DE, drug effects
Cell Membrane: EN, enzymology
Cell Membrane: ME, metabolism
Diffusion: DE, drug effects
Electric Conductivity
Fatty Acids, Unsaturated: CH, chemistry
***Fatty Acids, Unsaturated: ME, metabolism**
Fatty Acids, Unsaturated: PD, pharmacology

Ligands
Mice
Oocytes: DE, drug effects
Oocytes: ME, metabolism
Rats
Receptors, Cannabinoid
Receptors, Drug: AG,. . .

RN **404-86-4 (Capsaicin); 58493-49-5 (olvanil);** 94421-68-8 (anandamide)

CN 0 (Arachidonic Acids); 0 (**Fatty Acids,** Unsaturated); 0 (Ligands); 0 (Receptors, Cannabinoid); 0 (Receptors, Drug); 0 (**arvanil**); 0 (cannabinoid receptor CB2, rat); 0 (capsaicin receptor); EC 3.5. (Amidohydrolases); EC 3.5.1.- (**fatty-acid** amide hydrolase)

L12 ANSWER 4 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN

AB The present invention provides an **antitumor** pharmaceutical composition comprising a N-vanillyl **fatty acid** amide containing a saturated or unsatd. **fatty acid** residue containing 14 to 32 carbon atoms which is related to capsaicin. An **antitumor** pharmaceutical composition comprising a N-vanillyl **fatty acid** amide has a low side-effect and a high **antitumor** effect, in particular against **melanoma** and **leukemia,** and has a very low pungency, a stimulatory and a preinflammatory effect. For example, the reaction of 0.2309 g of vanillylamine with 0.5919 of 4,7,10,13,16,19-**docosahexaenoic** acid (C22:6, DHA) gave 0.311 g of colorless or citrine amorphous-like solid of N-vanillyl-4,7,10,13,16,19-**docosahexaenamide** (Dohevanyl). **Antitumor** effects of Dohevanyl were compared to those of capsaicin. Compared with capsaicin, Dohevanyl was very low in the degree of hotness and stimulus, and had a higher **antitumor** effect with a low action to the normal cells. Both capsaicin and Dohevanyl induced apoptosis to cause the cell death.

2004:470289 Document Number 141:17594 **Antitumor** pharmaceutical composition comprising N-vanillyl **fatty acid** amide.
Takahata, Kyoya (Kureha Chemical Industry Company, Limited, Japan). Eur. Pat. Appl. EP 1426047 A1 20040609, 22 pp. DESIGNATED STATES: R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK. (English). CODEN: EPXXDW. APPLICATION: EP 2003-254668 20030725. PRIORITY: JP 2002-353649 20021205.

TI **Antitumor** pharmaceutical composition comprising N-vanillyl **fatty acid** amide

AB The present invention provides an **antitumor** pharmaceutical composition comprising a N-vanillyl **fatty acid** amide containing a saturated or unsatd. **fatty acid** residue containing 14 to 32 carbon atoms which is related to capsaicin. An **antitumor** pharmaceutical composition comprising a N-vanillyl **fatty acid** amide has a low side-effect and a high **antitumor** effect, in particular against **melanoma** and **leukemia,** and has a very low pungency, a stimulatory and a preinflammatory effect. For example, the reaction of 0.2309 g of vanillylamine with 0.5919 of 4,7,10,13,16,19-**docosahexaenoic** acid (C22:6, DHA) gave 0.311 g of colorless or citrine amorphous-like solid of N-vanillyl-4,7,10,13,16,19-**docosahexaenamide** (Dohevanyl). **Antitumor** effects of Dohevanyl were compared to those of capsaicin. Compared with capsaicin, Dohevanyl was very low in the degree of hotness and stimulus, and had a higher **antitumor** effect with a low action to the normal cells. Both capsaicin and Dohevanyl induced apoptosis to cause the cell death.

ST vanillyl **fatty acid** amide prepn **antitumor**

IT Amides, biological studies
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(fatty; preparation of **antitumor** vanillyl **fatty acid** amides)

IT **Antitumor** agents
Apoptosis
Human
Leukemia
Melanoma
(preparation of **antitumor** vanillyl **fatty acid** amides)

IT **404-86-4**, Capsaicin
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); BIOL (Biological study)
(comparison with; preparation of **antitumor** vanillyl **fatty acid** amides)

IT **16729-47-8P**, N-Vanillyllinoleamide **58493-49-5P**, N-Vanillyloleamide **69693-12-5P**, N-Vanillylmyristamide **104899-01-6P 457643-60-6P**, N-Vanillylricinoleamide **571203-58-2P**, Dohevanil **698373-40-9P** **698373-42-1P**
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of **antitumor** vanillyl **fatty acid** amides)

IT 9001-62-1, Novozyme 435
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(preparation of **antitumor** vanillyl **fatty acid** amides)

IT 112-62-9, Methyl oleate 112-63-0, Methyl linoleate 124-10-7, Methyl myristate 6217-54-5 7149-10-2, Vanillylamine hydrochloride
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of **antitumor** vanillyl **fatty acid** amides)

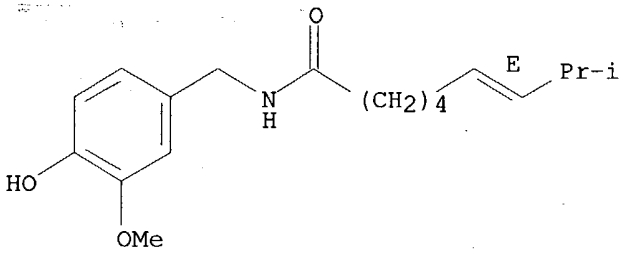
IT 1196-92-5P, Vanillylamine
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation of **antitumor** vanillyl **fatty acid** amides)

IT **404-86-4**, Capsaicin
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); BIOL (Biological study)
(comparison with; preparation of **antitumor** vanillyl **fatty acid** amides)

RN 404-86-4 HCAPLUS

CN 6-Nonenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-8-methyl-, (6E)- (9CI) (CA INDEX NAME)

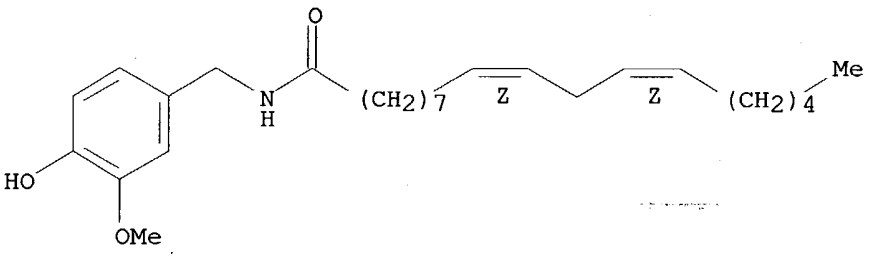
Double bond geometry as shown.



IT **16729-47-8P**, N-Vanillyllinoleamide **58493-49-5P**, N-Vanillyloleamide **69693-12-5P**, N-Vanillylmyristamide **104899-01-6P 457643-60-6P**, N-Vanillylricinoleamide **571203-58-2P**, Dohevanil **698373-40-9P** **698373-42-1P**
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of **antitumor** vanillyl **fatty acid** amides)

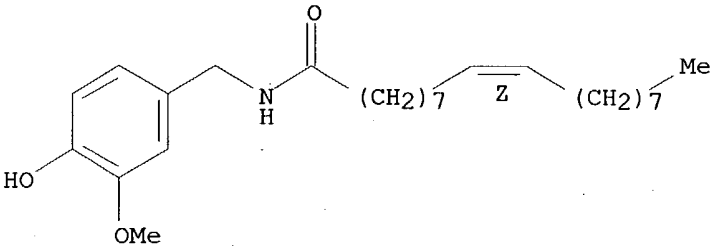
RN 16729-47-8 HCAPLUS
CN 9,12-Octadecadienamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



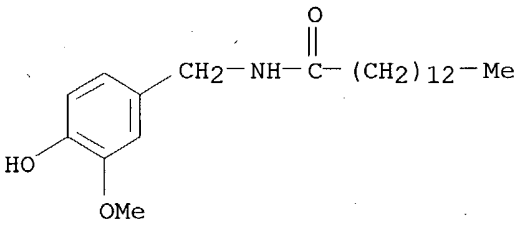
RN 58493-49-5 HCAPLUS
CN 9-Octadecenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (9Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 69693-12-5 HCAPLUS
CN Tetradecanamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]- (9CI) (CA INDEX

NAME)



RN 104899-01-6 HCAPLUS
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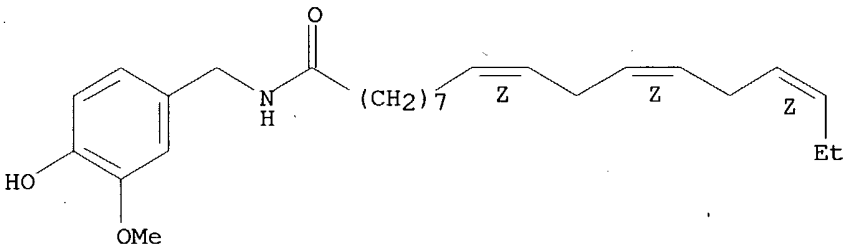
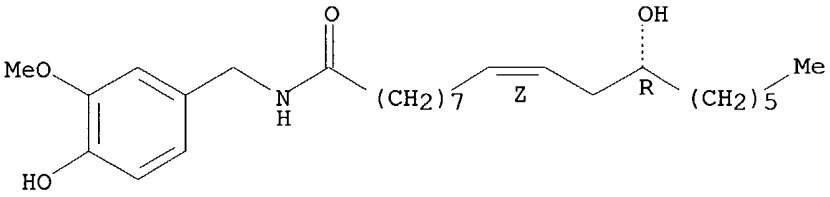
Double bond geometry as shown.



RN 457643-60-6 HCAPLUS
CN 9-Octadecenamide, 12-hydroxy-N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (9Z,12R)- (9CI) (CA INDEX NAME)

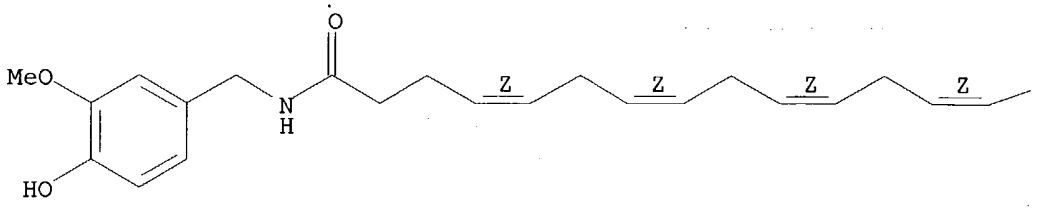
Absolute stereochemistry.
Double bond geometry as shown.



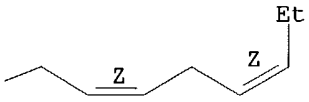
RN 571203-58-2 HCAPLUS
CN 4,7,10,13,16,19-Docosahexaenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (4Z,7Z,10Z,13Z,16Z,19Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



RN 698373-40-9 HCAPLUS
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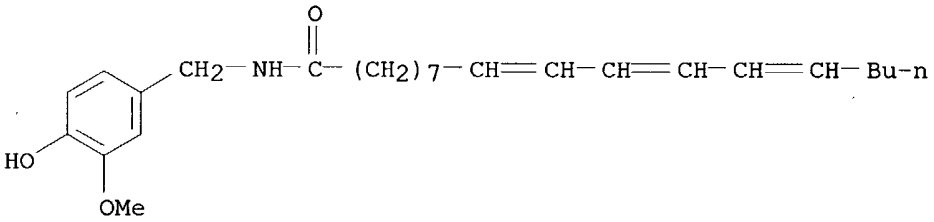
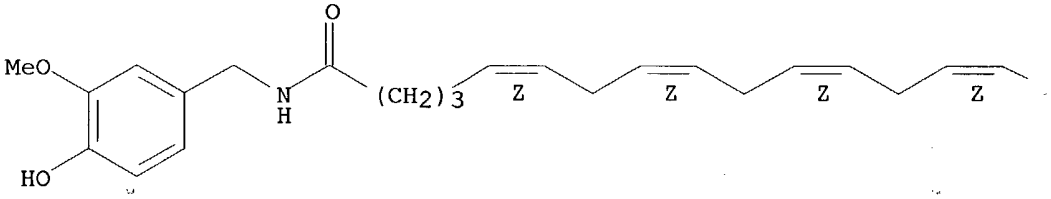


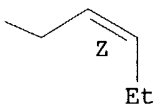
RN 698373-42-1 HCAPLUS
CN 5,8,11,14,17-Eicosapentaenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (5Z,8Z,11Z,14Z,17Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



DELACROIX

L12 ANSWER 5 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN

AB There are few effective clin. studies to inhibit the growth of multidrug resistance tumor cells. We have been interested in the physiol. actions of capsaicin (CAP), the pungent ingredient in hot chilli peppers, and polyunsatd. **fatty acids**, for example **docosahexaenoic** acid (DHA), extracted from fish oil. In this study, we synthesized a new vanillylamide derivative, N-**docosahexaenoylvanillylamide** (dohevanil), to investigate the inhibitory effect of dohevanil on growth of HeLa cells and taxol-tolerant HeLa cells. As a result, dohevanil has more potent inhibitory effect than CAP for both taxol-sensitive HeLa cells and taxol-tolerant HeLa cells. Particularly, the simultaneous addition of dohevanil and taxol more strongly induced cell death of taxol-tolerant HeLa cells. There results obtained in this study suggest that dohevanil has stronger inhibitory effect than CAP for the multidrug resistance cells.

2004:272650 Document Number 141:99178 Effect of capsaicin and N-**docosahexaenoyl**-vanillylamide on growth of taxol-tolerant HeLa cells. Jin, Yongfu; Ishihata, Kimie; Kajiyama, Shin-ichiro; Fukusaki, Ei-ichiro; Kobayashi, Akio; Baba, Naomichi; Tada, Mikiro; Takahata, Kyoya (Graduate School of Natural Science and Technology, Okayama University, Japan). Nippon Shokuhin Kagaku Gakkaishi, 9(2), 50-53 (Japanese) 2002. CODEN: NSKGF4. ISSN: 1341-2094. Publisher: Nippon Shokuhin Kagaku Gakkai.

TI Effect of capsaicin and N-**docosahexaenoyl**-vanillylamide on growth of taxol-tolerant HeLa cells

AB . . . We have been interested in the physiol. actions of capsaicin (CAP), the pungent ingredient in hot chilli peppers, and polyunsatd. **fatty acids**, for example **docosahexaenoic** acid (DHA), extracted from fish oil. In this study, we synthesized a new vanillylamide derivative, N-**docosahexaenoylvanillylamide** (dohevanil), to investigate the inhibitory effect of dohevanil on growth of HeLa cells and taxol-tolerant HeLa cells. As a result,. . .

ST capsaicin **docosahexaenoylvanillylamide** dohevanil taxol resistance tumor

IT **Antitumor** agents
Human
Multidrug resistance
(effect of capsaicin and N-**docosahexaenoyl**-vanillylamide on growth of taxol-tolerant HeLa cells)

IT Drug interactions
(synergistic; effect of capsaicin and N-**docosahexaenoyl**-vanillylamide on growth of taxol-tolerant HeLa cells)

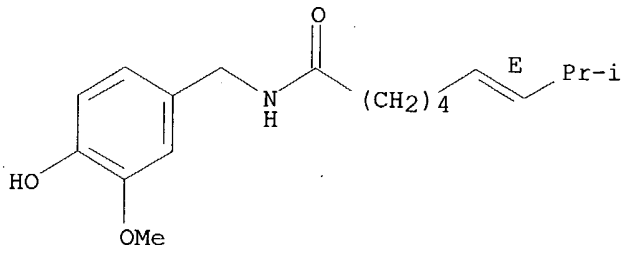
IT **404-86-4**, Capsaicin 33069-62-4, Taxol **571203-58-2**, Dohevanil
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(effect of capsaicin and N-**docosahexaenoyl**-vanillylamide on growth of taxol-tolerant HeLa cells)

IT **404-86-4**, Capsaicin **571203-58-2**, Dohevanil
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(effect of capsaicin and N-**docosahexaenoyl**-vanillylamide on growth of taxol-tolerant HeLa cells)

RN 404-86-4 HCAPLUS

CN 6-Nonenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-8-methyl-, (6E)- (9CI) (CA INDEX NAME)

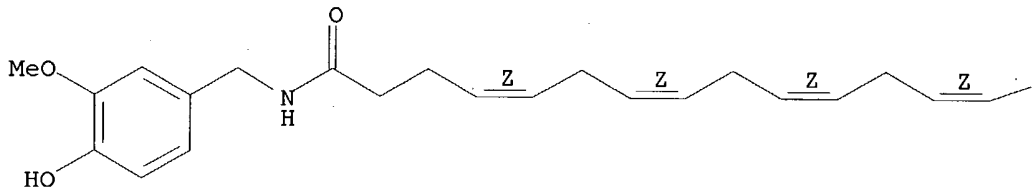
Double bond geometry as shown.



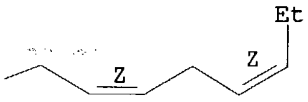
RN 571203-58-2 HCAPLUS
CN 4,7,10,13,16,19-Docosahexaenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (4Z,7Z,10Z,13Z,16Z,19Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



L12 ANSWER 6 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
AB **Arvanil**, a structural "hybrid" between the endogenous cannabinoid CB1 receptor ligand anandamide and capsaicin, is a potent agonist for the capsaicin receptor VR1 (vanilloid receptor type 1), inhibits the anandamide membrane transporter (AMT), and induces cannabimimetic responses in mice. Novel **arvanil** derivs. prepared by N-methylation, replacement of the amide with urea and thiourea moieties, and manipulation of the vanillyl group were evaluated for their ability to bind/activate CB1 receptors, activate VR1 receptors, inhibit the AMT and **fatty acid** amide hydrolase (FAAH), and produce cannabimimetic effects in mice. The compds. did not stimulate the CB1 receptor. Methylation of the amide group decreased the activity at VR1, AMT, and FAAH. On the aromatic ring, the substitution of the 3-methoxy group with a chlorine atom or the lack of the 4-hydroxy group decreased the activity on VR1 and AMT, but not the affinity for CB1 receptors, and increased the capability to inhibit FAAH. The urea or thiourea analogs retained activity at VR1 and AMT but exhibited little affinity for CB1 receptors. The urea analog was a potent FAAH inhibitor (IC50 = 2.0

μM). A water-soluble analog of **arvanil**, O-2142, was as active on VR1, much less active on AMT and CB1, and more potent on FAAH. All compds. induced a response in the mouse "tetrad", particularly those with EC50 <10 nM on VR1. However, the most potent compound, N-N'-di-(3-chloro-4-hydroxy)benzyl-arachidonamide (O-2093, ED50 .apprx.0.04 mg/kg), did not activate VR1 or CB1 receptors. Our findings suggest that VR1 and/or as yet uncharacterized receptors produce cannabimimetic responses in mice in vivo.

2002:203609 Document Number 137:56979 A structure/activity relationship study on **arvanil**, an endocannabinoid and vanilloid hybrid. Di Marzo, Vincenzo; Griffin, Graeme; De Petrocellis, Luciano; Brandi, Ines; Bisogno, Tiziana; Williams, William; Grier, Mark C.; Kulasegram, Sanjitha; Mahadevan, Anu; Razdan, Raj K.; Martin, Billy R. (Endocannabinoid Research Group, Istituto di Chimica Biomolecolare, Naples, Italy). Journal of Pharmacology and Experimental Therapeutics, 300(3), 984-991 (English) 2002. CODEN: JPETAB. ISSN: 0022-3565. OTHER SOURCES: CASREACT 137:56979. Publisher: American Society for Pharmacology and Experimental Therapeutics.

TI A structure/activity relationship study on **arvanil**, an endocannabinoid and vanilloid hybrid

AB **Arvanil**, a structural "hybrid" between the endogenous cannabinoid CB1 receptor ligand anandamide and capsaicin, is a potent agonist for the capsaicin receptor VR1 (vanilloid receptor type 1), inhibits the anandamide membrane transporter (AMT), and induces cannabimimetic responses in mice. Novel **arvanil** derivs. prepared by N-methylation, replacement of the amide with urea and thiourea moieties, and manipulation of the vanillyl group were evaluated for their ability to bind/activate CB1 receptors, activate VR1 receptors, inhibit the AMT and **fatty acid** amide hydrolase (FAAH), and produce cannabimimetic effects in mice. The compds. did not stimulate the CB1 receptor. Methylation of the. . . affinity for CB1 receptors. The urea analog was a potent FAAH inhibitor (IC50 = 2.0 μM). A water-soluble analog of **arvanil**, O-2142, was as active on VR1, much less active on AMT and CB1, and more potent on FAAH. All compds.. . .

ST **arvanil** deriv cannabinoid receptor binding structure design analgesic

IT Capsaicin receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(VR1; structure/activity relationship study on **arvanil**)

IT Structure-activity relationship
(analgesic; structure/activity relationship study on **arvanil**)

IT Structure-activity relationship
(hypotensive; structure/activity relationship study on **arvanil**)

IT Drug delivery systems
(injections, i.v.; structure/activity relationship study on **arvanil**)

IT Behavior
(locomotor; structure/activity relationship study on **arvanil**)

IT Structure-activity relationship
(receptor-binding, cannabinoid; structure/activity relationship study on **arvanil**)

IT Body temperature
(rectal; structure/activity relationship study on **arvanil**)

IT Amide group
Anti-inflammatory agents
Antitumor agents

Drug design
Hydroxyl group
Methoxy group
(structure/activity relationship study on **arvanil**)

IT Cannabinoid receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type CB1; structure/activity relationship study on **arvanil**)

IT 7440-70-2, Calcium, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cytosolic; structure/activity relationship study on **arvanil**)

IT 153301-19-0, **Fatty acid** amide hydrolase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(structure/activity relationship study on **arvanil**)

IT 57-13-6, Urea, biological studies 62-56-6, Thiourea, biological studies
RL: BSU (Biological study, unclassified); RCT (Reactant); BIOL (Biological study); RACT (Reactant or reagent)
(structure/activity relationship study on **arvanil**)

IT **322399-59-7P**, O-1861 439079-98-8P, O 1988 439079-99-9P, O 1986 439080-00-9P, O 2094 439080-01-0P, O 2093 439080-02-1P, O 1987 439080-03-2P 439080-04-3P, O 2109 439080-05-4P, O 2142
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(structure/activity relationship study on **arvanil**)

IT **128007-31-8P, Arvanil**
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
(structure/activity relationship study on **arvanil**)

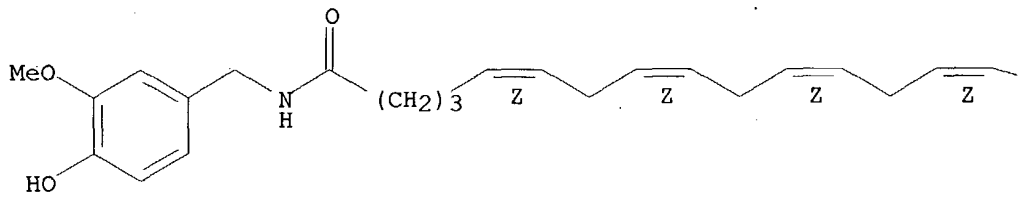
IT 121-33-5, 3-Methoxy-4-hydroxybenzaldehyde **404-86-4**, Capsaicin 506-32-1, Arachidonic acid 2420-16-8, 3-Chloro-4-hydroxybenzaldehyde 5807-09-0, 4-Morpholinebutanoic acid 22537-15-1, Chlorine atom, reactions 57303-04-5, Arachidonic acid chloride 94421-68-8, Anandamide 184003-34-7 366825-49-2 438449-98-0 438449-99-1
RL: RCT (Reactant); RACT (Reactant or reagent)
(structure/activity relationship study on **arvanil**)

IT **322399-59-7P**, O-1861
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(structure/activity relationship study on **arvanil**)

RN 322399-59-7 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, 20-bromo-N-[(4-hydroxy-3-methoxyphenyl)methyl]-16,16-dimethyl-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

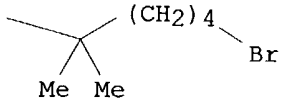
Double bond geometry as shown.

PAGE 1-A



DELACROIX

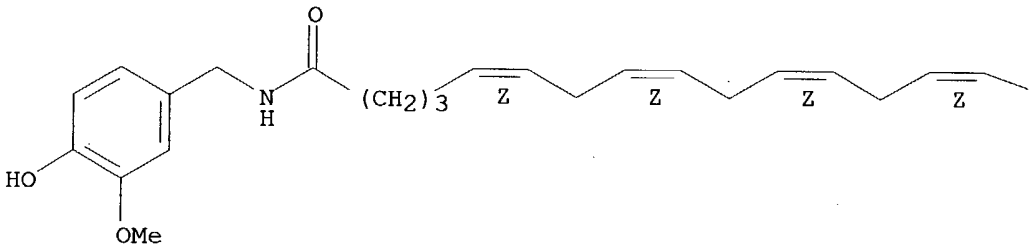
PAGE 1-B



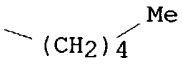
IT **128007-31-8P, Arvanil**
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
(structure/activity relationship study on **arvanil**)
RN 128007-31-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A

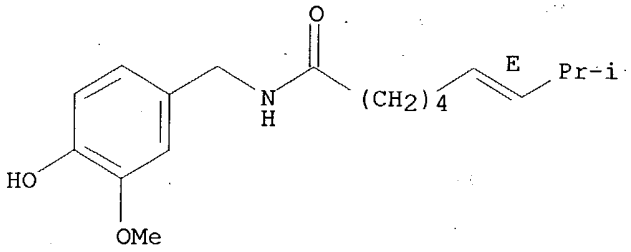


PAGE 1-B



IT **404-86-4**, Capsaicin
RL: RCT (Reactant); RACT (Reactant or reagent)
(structure/activity relationship study on **arvanil**)
RN 404-86-4 HCAPLUS
CN 6-Nonenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-8-methyl-, (6E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L12 ANSWER 7 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN

AB The endogenous cannabinoid receptor agonist anandamide (AEA) and the related compound palmitoylethanolamide (PEA) are inactivated by transport into cells followed by metabolism by **fatty acid** amide hydrolase (FAAH). The cellular uptake of AEA has been characterized in detail, whereas less is known about the properties of the PEA uptake, in particular in neuronal cells. In the present study, the pharmacol. and functional properties of PEA and AEA uptake have been investigated in mouse Neuro-2a neuroblastoma and, for comparison, in rat RBL-2H3 basophilic **leukemia** cells. Saturable uptake of PEA and AEA into both cell lines were demonstrated with apparent KM values of 28 μM (PEA) and 10 μM (AEA) in Neuro-2a cells, and 30 μM (PEA) and 9.3 μM (AEA) in RBL-2H3 cells. Both PEA and AEA uptake showed temperature-dependence but only the AEA uptake was sensitive to treatment with Pronase and phenylmethylsulfonyl fluoride. The AEA uptake was inhibited by AM404, 2-arachidonoylglycerol (2-AG), R1- and S1-methanandamide, arachidonic acid and **olvanil** with similar potencies for the two cell types. PEA, up to a concentration of 100 μM, did not affect AEA uptake in either cell line. AEA, 2-AG, arachidonic acid, R1-methanandamide, Δ9-THC, and cannabidiol inhibited PEA transport in both cell lines. The non-steroidal anti-inflammatory drug indomethacin inhibited the AEA uptake but had very weak effects on the uptake of PEA. From these data, it can be concluded that PEA is transported in to cells both by passive diffusion and by a facilitated transport that is pharmacol. distinguishable from AEA uptake.

2001:322837 Document Number 135:132395 Characterization of palmitoylethanolamide transport in mouse Neuro-2a neuroblastoma and rat RBL-2H3 basophilic leukaemia cells: comparison with anandamide. Jacobsson, Stig O. P.; Fowler, Christopher J. (Department of Pharmacology and Clinical Neuroscience, Department of Odontology, Umea University, Umea, SE-901 87, Swed.). British Journal of Pharmacology, 132(8), 1743-1754 (English) 2001. CODEN: BJPCBM. ISSN: 0007-1188. Publisher: Nature Publishing Group.

AB . . . receptor agonist anandamide (AEA) and the related compound palmitoylethanolamide (PEA) are inactivated by transport into cells followed by metabolism by **fatty acid** amide hydrolase (FAAH). The cellular uptake of AEA has been characterized in detail, whereas less is known about the properties. . . properties of PEA and AEA uptake have been investigated in mouse Neuro-2a neuroblastoma and, for comparison, in rat RBL-2H3 basophilic **leukemia** cells. Saturable uptake of PEA and AEA into both cell lines were demonstrated with apparent KM values of 28 μM. . . with Pronase and phenylmethylsulfonyl fluoride. The AEA uptake was inhibited by AM404, 2-arachidonoylglycerol (2-AG), R1- and S1-methanandamide, arachidonic acid and **olvanil** with similar potencies for the two cell types. PEA, up to a concentration of 100

μM, did not affect AEA. . .

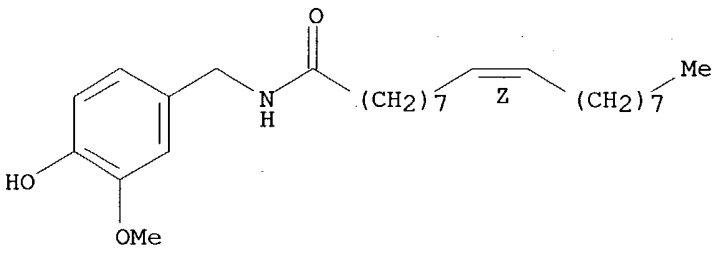
IT 53-86-1, Indomethacin 329-98-6, Phenylmethylsulfonyl fluoride
506-32-1, Arachidonic acid 1972-08-3, Δ9-THC 9036-06-0, Pronase
13956-29-1, Cannabidiol 15687-27-1, Ibuprofen 53847-30-6
58493-49-5, Olvanil 157182-49-5, R-Methanandamide
157182-50-8, S-Methanandamide
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(pharmacol. characterization of palmitoylethanolamide transport in neuronal cells)

IT **58493-49-5, Olvanil**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(pharmacol. characterization of palmitoylethanolamide transport in neuronal cells)

RN 58493-49-5 HCAPLUS

CN 9-Octadecenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (9Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L12 ANSWER 8 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

AB On the basis of temperature dependency, saturability, selective inhibition, and substrate specificity, it has been proposed that an anandamide transporter exists. However, all of these studies have examined anandamide accumulation at long time points when downstream effects such as metabolism and intracellular sequestration are operative. In the current study, we have investigated the initial rates (<1 min) of anandamide accumulation in neuroblastoma and astrocytoma cells in culture and have determined that uptake is not saturable with increasing concentrations of anandamide. However, anandamide hydrolysis, after uptake in neuroblastoma cells, was saturable at steady-state time points (5 min), suggesting that **fatty acid** amide hydrolase (FAAH) may be responsible for observed saturation of uptake at long time points. In general, **arvanil, olvanil,** and N-(4-hydroxyphenyl)arachidonylamide (AM404) have been characterized as transport inhibitors in studies using long incubations. However, we found these "transport inhibitors" did not inhibit anandamide uptake in neuroblastoma and astrocytoma cells at short time points (40 sec or less). Furthermore, we confirmed that these inhibitors in vitro were actually inhibitors of FAAH. Therefore, the likely mechanism by which the transport inhibitors raise anandamide levels to exert pharmacological effects is by inhibiting FAAH, and they should be reevaluated in this context. Immunofluorescence has indicated that FAAH staining resides mainly on intracellular membranes of neuroblastoma cells, and this finding

is consistent with our observed kinetics of anandamide hydrolysis. In summary, these data suggest that anandamide uptake is a process of simple diffusion. This process is driven by metabolism and other downstream events, rather than by a specific membrane-associated anandamide carrier.

2003:252331 Document Number: PREV200300252331. Evidence against the presence of an anandamide transporter. Glaser, Sherrye T.; Abumrad, Nada A.; Fatade, Folayan; Kaczocha, Martin; Studholme, Keith M.; Deutsch, Dale G. [Reprint Author]. Department of Biochemistry and Cell Biology, Stony Brook University, Stony Brook, NY, 11794, USA. ddeutsch@notes.cc.sunysb.edu. Proceedings of the National Academy of Sciences of the United States of America, (April 1 2003) Volume 100, Number 7, pp. 4269-4274. print. ISSN: 0027-8424 (ISSN print). Language: English.

AB. . . of anandamide. However, anandamide hydrolysis, after uptake in neuroblastoma cells, was saturable at steady-state time points (5 min), suggesting that **fatty acid** amide hydrolase (FAAH) may be responsible for observed saturation of uptake at long time points. In general, **arvanil**, **olvanil**, and N-(4-hydroxyphenyl)arachidonylamide (AM404) have been characterized as transport inhibitors in studies using long incubations. However, we found these "transport inhibitors". . .

IT . . . Concepts
Biochemistry and Molecular Biophysics; Membranes (Cell Biology)

IT Parts, Structures, & Systems of Organisms
cell; membrane

IT Diseases
astrocytoma: **neoplastic** disease, nervous system disease
Astrocytoma (MeSH)

IT Diseases
neuroblastoma: **neoplastic** disease, nervous system disease
Neuroblastoma (MeSH)

IT Chemicals & Biochemicals
AM404: enzyme inhibitor-drug; anandamide; anandamide transporter; **arvanil**: enzyme inhibitor-drug; **fatty acid** amide hydrolase; **olvanil**: enzyme inhibitor-drug

RN 183718-77-6 (AM404)
94421-68-8 (anandamide)
128007-31-8 (**arvanil**)
153301-19-0 (**fatty acid** amide hydrolase)
58493-49-5 (**olvanil**)

L12 ANSWER 9 OF 9 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

AB The long history of the medicinal use of Cannabis sativa and, more recently, of its chemical constituents, the cannabinoids, suggests that also the endogenous ligands of cannabinoid receptors, the endocannabinoids, and, particularly, their derivatives may be used as therapeutic agents. Studies aimed at correlating the tissue and body fluid levels of endogenous cannabinoid-like molecules with pathological conditions have been started and may lead to identify those diseases that can be alleviated by drugs that either mimic or antagonize the action of these substances, or modulate their biosynthesis and degradation. Hints for the therapeutic applications of endocannabinoids, however, can be obtained also from our previous knowledge of marijuana medicinal properties. In this article, we discuss the **anti-tumor** and **anti**-inflammatory activity of: (1) the endocannabinoids anandamide (arachidonoylethanolamide) and 2-arachidonoyl glycerol; (2) the bioactive **fatty acid** amides palmitoylethanolamide and

oleamide; and (3) some synthetic derivatives of these compounds, such as the N-acyl-vanillyl-amines. Furthermore, the possible role of cannabimimetic **fatty acid** derivatives in the pathological consequences of **cancer** and inflammation, such as cachexia, wasting syndrome, chronic pain and local vasodilation, will be examined. (C) 2000 Elsevier Science Ireland Ltd.

2000425328 EMBASE Endocannabinoids and **fatty acid** amides in **cancer**, inflammation and related disorders. De Petrocellis L.; Melck D.; Bisogno T.; Di Marzo V.. V. Di Marzo, Ist. Chimica Molecole Int. Biologico, C.N.R., via Toiano 6, 80072 Arco Felice, Napoli, Italy. vdimarzo@icmib.na.cnr.it. Chemistry and Physics of Lipids 108/1-2 (191-209) 2000.
Refs: 104.
ISSN: 0009-3084. CODEN: CPLIA4.
Publisher Ident.: S 0009-3084(00)00196-1. Pub. Country: Ireland. Language: English. Summary Language: English.

TI Endocannabinoids and **fatty acid** amides in **cancer**, inflammation and related disorders.

AB . . . endocannabinoids, however, can be obtained also from our previous knowledge of marijuana medicinal properties. In this article, we discuss the **anti-tumor** and **anti**-inflammatory activity of: (1) the endocannabinoids anandamide (arachidonoylethanolamide) and 2-arachidonoyl glycerol; (2) the bioactive **fatty acid** amides palmitoylethanolamide and oleamide; and (3) some synthetic derivatives of these compounds, such as the N-acyl-vanillyl-amines. Furthermore, the possible role of cannabimimetic **fatty acid** derivatives in the pathological consequences of **cancer** and inflammation, such as cachexia, wasting syndrome, chronic pain and local vasodilation, will be examined. (C) 2000 Elsevier Science Ireland. . .

CT Medical Descriptors:
***cancer: DT, drug therapy**
*inflammation: DT, drug therapy
*chronic pain: CO, complication
*chronic pain: DT, drug therapy
*chronic pain: ET, etiology
*wasting syndrome: CO, complication
*wasting syndrome:. . . activity
food intake
drug indication
drug efficacy
treatment outcome
human
nonhuman
article
priority journal
*cannabinoid: CB, drug combination
*cannabinoid: CM, drug comparison
*cannabinoid: DT, drug therapy
*cannabinoid: EC, endogenous compound
*cannabinoid: PD, pharmacology
***fatty acid derivative: DT, drug therapy**
***fatty acid derivative: PD, pharmacology**
*amine: EC, endogenous compound
*amine: PD, pharmacology
cannabis: DT, drug therapy
cannabis: PD, pharmacology

cannabinoid receptor: EC, endogenous compound
anandamide: CB, drug. . . therapy
n acylvanillylamine derivative: EC, endogenous compound
n acylvanillylamine derivative: PD, pharmacology
di homo gamma linolenoylethanolamide: DT, drug therapy
di homo gamma linolenoylethanolamide: PD, pharmacology
docosahexanoylethanolamide: DT, drug therapy
docosahexanoylethanolamide: PD, pharmacology
tetrahydrocannabinol: DT, drug therapy
tetrahydrocannabinol: PD, pharmacology
5 (4 chlorophenyl) 1 (2,4 dichlorophenyl) 4 methyl n (1 piperidyl) 1h pyrazole 3. . . 5 methyl 3 (morpholinomethyl) 6 (1 naphthoyl)pyrrolo[1,2,3 de][1,4]benzoxazine: PD, pharmacology
4 (1,1 dimethylheptyl) 1',2',3',4',5',6' hexahydro 2,3' dihydroxy 6' (3 hydroxypropyl)biphenyl: PD, pharmacology
arvanil: PD, pharmacology
olvanil: PD, pharmacology
linvanil: PD, pharmacology
n arachidonoyldopamine: PD, pharmacology
bml 190: CB, drug combination
bml 190: CM, drug comparison
bml 190: PD, pharmacology
unclassified drug

RN. . . 5 methyl 3 (morpholinomethyl) 6 (1 naphthoyl)pyrrolo[1,2,3 de][1,4]benzoxazine) 134959-51-6; (4 (1,1 dimethylheptyl) 1',2',3',4',5',6' hexahydro 2,3' dihydroxy 6' (3 hydroxypropyl)biphenyl) 83003-12-7; (**olvanil**) **58493-49-5**